THE Q-T INTERVAL IN RHEUMATIC FEVER

BY

J. N. BRIGGS and S. A. DOXIADIS

From the Department of Child Health, University of Sheffield

(RECEIVED FOR PUBLICATION JANUARY 13, 1951)*

The prolongation of the Q-T interval in the electrocardiogram of patients suffering from valvular disease or rheumatic carditis was noticed by Berliner (1931) and by Drawe, Hafkesbring, and Ashman (1937). These observations were not considered to be of value for the diagnosis of cardiac involvement during or after an attack of acute rheumatic fever until Taran and Szilagyi (1947), studying a series of 50 cases, claimed that the duration of electrical systole was lengthened in all children whose electrocardiograms had been taken during an attack of acute rheumatic carditis.

Their report revived interest in the value of the measurement of the Q-T interval, and further papers have since been published: Abrahams (1949) found a prolonged Q-T interval in 90 of his 100 patients with active carditis, and agreed with Taran and Szilagyi (1947) that this measurement was of diagnostic value. However in Pokress' and Goldberger's (1949) series only 14 of the 50 patients (28%) with active rheumatic fever had a Q-T interval above the normal maximal value.

The results of the present series differ significantly from those of Taran and Szilagyi (1947) and of Abrahams (1949). This paper* is an attempt to explain this difference, and to assess the value of the measurement of the Q-T interval in rheumatic fever.

Methods

Goldberger's (1948) nomogram was chosen to relate the measured Q-T interval to the heart rate. This nomogram expresses the Q-T interval as the ratio of the measured to the ideal interval for that heart rate. The ideal Q-T interval for each heart rate is estimated by the use of Bazett's (1920) formula, and since this formula was used in the other reports, we were able to compare our results with those of the earlier workers.

All recordings were made with the child in a reclining posture, and after he had been in bed for at least 24 hours.

In approximately half the cases the apparatus used was the General Electric Company electrocardiographic machine, and in the rest the Elmquist Junior (General Radiological Company) machine.

Lead II was used for the measurements, unless the end of the T wave was ill defined, in which case the complexes in lead I or lead III were preferred. The measurements were made with a pair of compass dividers and a hand lens. Particular care was taken while measuring the Q-T interval to measure it from the beginning of the Q deviation to the point at which the T wave again became isoelectric. The Q-T and R-R intervals of at least five cardiac cycles were measured, and the Q-T ratio of each separate cycle was found on the nomogram and the average of these values estimated.

Material

One hundred and twelve electrocardiograms of 102 children, whose ages ranged from $3\frac{1}{2}$ to 14 years, were examined. All the children were in-patients at the Children's Hospital, Sheffield, and at the Ash House Hospital School. Eighty-two had rheumatic fever at various stages, and the remaining 20 were controls. They were divided into the following groups.

Group I. These children had definite evidence of active rheumatic infection. Polyarthritides, marked lassitude, and anorexia were the main features of their present illness. The sedimentation rate estimated on admission by the micromethod was above 20 mm. in one hour, and remained elevated for at least six weeks, in the absence of other discoverable causes. At the time the electrocardiogram was recorded, polyarthritides had subsided in most of the children but lassitude, pallor, and anorexia persisted, while the sedimentation rate remained high. This group was further subdivided into two sub-groups, Ia and Ib. The children in sub-group Ia either had at the time of the recording, or developed within a short time after the recording and during the same attack of rheumatic fever, an apical systolic murmur of moderate or loud intensity (Levine's (1933) grade III to grade VI), harsh in

* Since this paper has been submitted for publication, two more articles on this subject have come to our notice. Craig, Alimurung, Blood, and Massull (1950), in an observation of 143 rheumatic children, have presented results which are closely similar to our own, and in a recent paper Taran and Szilagyi (1951) claim further supporting evidence for their earlier work.

FIG. 1.—Q-T ratios in children with rheumatic infection and in controls.

nature, long, and well conducted to the axilla, or an apical diastolic murmur, or both. These auscultatory findings were always confirmed by more than one observer. In none of these cases was a friction rub heard at any time.

The children in sub-group Ib neither had, nor subsequently developed during the same attack of rheumatic fever, any of the murmurs described above.

Group II. This consisted of children who were known to have had an attack of acute rheumatic fever with carditis, as defined by the criteria laid down for sub-group Ia, and in whom one or both of the murmurs described persisted. All clinical signs of activity had, however, subsided at the time of the recording. Only children whose weekly sedimentation rate had been below 10 mm. in one hour for at least two months were included in this group.

Group III. The controls were children of the same age group who were admitted for minor surgical operations, and had neither signs of infection nor abnormal cardiac signs.

The number of children and of recordings in each group is shown in Table 1.

TABLE I
NUMBER OF CHILDREN AND RECORDINGS IN THAT GROUP

Group	Number of Children	E.C.G. Recordings
Ia. Active rheumatic fever ; carditis definite ..	46	55
Ib. Active rheumatic fever ; carditis doubtful ..	12	13
II. Inactive rheumatic heart disease	24	24
III. Normal controls ..	20	20
Total	102	112

Results

Individual Measurements. In Fig. 1 the Q-T ratios for the four groups are presented. The lower horizontal line at 1.01 corresponds roughly to a Q-T interval of 0.405 corrected according to Bazett's (1920) formula. It represents the upper limit of normal in Taran's, and Szilagyi's (1947) series. The upper horizontal line at 1.06 corresponds to a corrected Q-T interval of 0.422 which represents the upper limit of normal Q-T interval for children (Ashman and Hull, 1937). This upper limit of normal has been also adopted by Abrahams

(1949), while Pokress and Goldberger (1949) place the upper limit of normal even higher, at 1.08.

It can be seen from Fig. 1 that the Q-T intervals of normal children were all below Ashman's and Hull's upper limit of normal, and this maximal normal value can therefore be accepted for the present study. Six recordings in Group Ia, two in Group Ib, and three in Group II showed a Q-T interval above the upper limit of normal.

Statistical Comparison. The figures of each group as a whole were compared with those of the other groups, and a difference was accepted as statistically significant when the possibility of its being due to chance was less than 5%. In these children in Group I of whom more than one recording was taken, the mean of the measurements in each child was used in the statistical comparison. There was no statistically significant difference between Group Ia and Group II, namely between children with active and inactive carditis, or between Group I as a whole (Ia and Ib) and Group II. Group I was not significantly different from the control Group III, while there was a significant difference between Groups II and III. Finally all the children with active rheumatic fever or inactive carditis taken together (Group I and II) had a Q-T interval significantly prolonged when compared to the Q-T interval of the 20 controls (Group III).

Quiescence. Five of the children in Group Ia eventually became inactive by our criteria. The Q-T intervals of two of these children were longer in the active stage.

Discussion

The value of the measurement of the Q-T interval in rheumatic fever must be assessed from two different angles. The first, of great practical importance, is whether the measurement helps in the diagnosis of active rheumatic carditis. The second, of more theoretical interest, is whether rheumatic infection exerts any influence at all on the duration of the electrical systole.

In only six of 55 recordings taken during an active rheumatic carditis (sub-group Ia) was the Q-T interval above the maximal normal limit. If to this number are added the children with rheumatic activity, but without definite cardiac involvement, the proportion of lengthened Q-T intervals becomes eight of 68 (11.8%). Three out of 24 children with inactive rheumatic carditis (Group II) had a prolonged Q-T interval (12%). These results suggest that in a case of rheumatic fever a Q-T interval above the normal limit indicates the presence of active rheumatic carditis, but this diagnostic help is available in only a small number of children.

It is evident also that the measurement of the Q-T interval does not help in deciding that rheumatic activity has ceased, since it was prolonged even in cases of inactive rheumatic heart disease.

It may be argued that in the three children in Group II with prolonged Q-T interval there was still latent rheumatic activity. This is unlikely, however, since these three children were allowed gradually to resume their normal life, and there was no evidence of reactivation of the rheumatic fever by the criteria defined.

These results are very different from those of Taran and Szilagyi (1947) and of Abrahams (1949). The former found that 100% of their cases with active rheumatic carditis had a Q-T interval longer than that found in their inactive ones. Unfortunately they do not state in their paper the criteria for the diagnosis of active carditis. In another publication, however, the senior author (Taran, 1947) states that one of the primary clinical criteria of active carditis is 'the disturbance in relationship of systole to diastole.' If such a criterion was indeed used for the selection of cases with active carditis, it is not surprising that all had a lengthened Q-T interval.

Abrahams (1949), on the other hand, defined more clearly the criteria for dividing his cases into active and inactive carditis, and these criteria are similar to those adopted in the present study. It is difficult, therefore, to determine why our findings are so different from his. In Abrahams' (1949) series the Q-T intervals are on the whole longer than our own, and it is unfortunate that in his paper neither the number of normal subjects examined nor the measurements of their Q-T intervals are given. Even from Abrahams' (1949) data it does not appear that the Q-T interval may help in establishing cessation of rheumatic activity, since in five out of 12 cases of inactive carditis the Q-T interval was lengthened. It is true that it was suspected that these five cases were not really inactive, but the reasons for suspecting this inactivity are given for one case only. It seems, therefore, that although rheumatic infection may prolong the duration of electrical systole, the change is so inconstant and slight that it is not often of diagnostic value. Even when it is, it may indicate the presence

of rheumatic carditis, but not whether the carditis is active or inactive.

Taran and Szilagyi (1947), and Abrahams (1949) have claimed that repeated measurements of the Q-T interval in the same child indicate a relationship between the duration of the electrical systole and the degree of rheumatic activity. The number of similar cases studied in the present series is too small to justify general conclusions. However, it must be noted that even among the electrocardiograms of five children, two did not demonstrate this relationship.

Summary

The Q-T interval was measured in 112 electrocardiograms of 82 children with rheumatic fever and of 20 normal controls.

The Q-T interval was above the maximal normal limit in eight of 68 tracings taken during active rheumatic infection, in three of 24 tracings of children with inactive rheumatic heart disease, and in none of the tracings obtained from the controls.

There was a significant difference between the mean Q-T interval of all the children with active or inactive carditis and the control group.

It is concluded that rheumatic infection may prolong the duration of the electrical systole, but the degree of this change is such that it is of diagnostic value in only a few cases.

We wish to thank Professor E. J. Wayne and Dr. T. Colver for permission to study their cases, Professor R. S. Illingworth for his advice, and Mrs. B. Clapham for statistical help.

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